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Oil Sands Contaminants

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In the article "Alberta's Oil Sands: Hard Evidence, Missing Data, New Promises," Weinhold (2011) misrepresented the findings of our Royal Society of Canada report (Gosselin et al. 2010) too often to recount fully here. Despite requesting my review of a draft, *EHP* chose not to correct many errors I identified, raising questions about *EHP*'s editorial bias on this matter. A few examples must suffice.

Apparently determined to find oil sands–related air quality problems beyond the odor issues we highlighted, Weinhold extracted data from our table summarizing 11 years of regional air quality monitoring data (Gosselin et al. 2010) to conclude:

> $PM_{2.5}$ exceedances at Fort McKay have been more than double those at the village of Anzac. … As anecdotal evidence of potential particulate matter concerns, a panel commissioned by Environment Canada to evaluate the impacts of oil sands operations referred to the "ubiquitous dust" that was present during their site visits.

Weinhold failed to explain that the Fort McKay site exceeded the 24 hr objective for $PM_{2.5}$ (30 μg/m^3) only nine times in 11 years, compared with Anzac at four times in 11 years. The implication that Fort McKay is suffering from oil sands $PM_{2.5}$ air pollution is inaccurate. Fort McKay is a rural northern community surrounded by oil sands surface mines, with local domestic combustion sources and occasional impact from regional forest fire smoke. Weinhold's attempt to validate his oil sands–related $PM_{2.5}$ pollution case by referring to anecdotal comments about "ubiquitous dust" near Fort McKay reflects his ignorance about what $PM_{2.5}$ measures, because it does not represent visible "dust."

Apparently searching for other air quality problems, Weinhold paraphrased our report to state: "There are more than 1,400 known pollutants emitted by oil sands operations." This was based on an inventory of all possible pollutants for developing air monitoring priorities. Weinhold neglected to include our next sentence: "The majority of the total mass emissions (98%) are made up of only fifteen compounds." But more important, any trace air contaminant expert can verify that thousands of pollutants can be found in any major urban area given sufficiently sensitive analytical techniques. No jurisdiction has air quality criteria for these countless trace substances. Weinhold's attempted revelation about oil sands contaminants being ignored lacks any meaningful air quality context.

Weinhold and *EHP* also chose not to correct his statements, which he directly attributed to our report:

> Studies have found that many toxics … can occur at higher concentrations downstream of oil sands operations than upstream (in some cases all the way to Lake Athabasca), and some of these are elevated enough to kill fish.

We advised *EHP* that we reported no evidence of higher levels of contaminants persisting to Lake Athabasca, nor did Weinhold's blanket statement about levels being elevated enough to kill fish accurately reflect our conclusions.

Another example of bias in the article appears in the caption of a photograph showing a Fort Chipewyan woman in a cemetery; the caption mentions our panel finding that evidence did not support a link between cancers in that community and oil sands contaminants, while noting that we recommended additional monitoring, but there is no mention that our additional monitoring proposal was made specifically to deal with community concerns. The caption continues: "That leaves this Fort Chipewyan woman still uncertain over what caused the lung cancer that killed her mother, husband, and 27-year-old nephew between 2006 and 2008." Using this emotive photo surely stoops below the standards of an unbiased scientific journal even if it had acknowledged the overwhelming cause of lung cancer. Readers need to know that extensive air quality monitoring in Fort Chipewyan has shown consistently excellent air quality, which has been verified by personal exposure studies. Regardless, it is crude sensationalism to imply that the personal tragedy depicted in this photo is relevant to cancer being caused by environmental contaminants.

Clearly, Weinhold is entitled to disagree with our panel's findings, particularly if he is writing an opinion piece. However, it is totally unacceptable for *EHP* to allow him to misinterpret extracts from our report and represent them in his article as if they were our findings. This is particularly egregious when the editors have been informed before publication of these misinterpretations.

In closing, I am compelled to forewarn any future national academy panel that may communicate with *EHP* having any expectation of it being an unbiased, objective scientific journal. *EHP* has behaved no better than agenda-driven commercial media that seek to spin their points of view regardless of the science.

The author declares that he has no actual or potential competing financial interests.

Steve E. Hrudey
Professor Emeritus
Analytical & Environmental Toxicology Division
Faculty of Medicine & Dentistry
University of Alberta
Edmonton, Alberta, Canada
E-mail: steve.hrudey@ualberta.ca

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Oil Sands Contaminants: Editor's Response

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The article by Weinhold (2011) offered an overview of potential environmental and health issues related to oil sands operations and was never intended to be nor presented solely as a recapitulation of the Royal Society of Canada (RSC) report (Gosselin et al. 2010). Instead, it presented a discussion of the environmental health information in that report as well as the related significant source documents it reviewed.

The information presented by Weinhold (2011) went beyond the conclusions in the RSC report in order to highlight the data used to reach those conclusions. It also provided information from a number of other sources, which at times conflicted with the RSC's conclusions even as it agreed with details in the report.

Both the RSC report and other reports document a range of health and environmental concerns in the Alberta oil sands operations area and beyond. Weinhold's article (2011) reflected that evidence and included numerous qualifying statements stipulating that many unknowns remain. The fledgling evidence, combined with major gaps in existing environmental health science and the fact that very little of the expected oil sands development has occurred, suggest that significant additional adverse effects cannot be ruled out as development expands. Given these facts, it would have been irresponsible journalism for Weinhold to have given oil sands operations an essentially clean bill of health.

The photograph on p. A130 of Weinhold's article (2011) speaks to the reality that many citizens of Fort Chipewyan continue to be concerned about the possible effects of oil sands activity on their health

and are uncertain about why community members are dying from what appear to the survivors to be unusual causes. Although Hrudey may feel this concern is misplaced or unfounded, that opinion does not reflect the feelings of those who live in Fort Chipewyan. The Alberta government's assertion that more extensive health studies are warranted (Chen 2009) and stated intention to actively pursue such studies (Weinhold 2011) suggest adverse health effects are at least plausible.

Bob Weinhold
Freelance Science Journalist
Colorado City, Colorado

Susan M. Booker
News Editor, *EHP*
Research Triangle Park, North Carolina
E-mail: booker@niehs.nih.gov

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Neurotoxicity of PBDEs on the Developing Nervous System

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Dingemans et al. (2011) published a review article on polybrominated diphenyl ethers (PBDEs) and the developing nervous system. However, the authors summarized but failed to critically evaluate the articles cited in their review. They also did not discuss or cite literature that contradicted the studies on which they based their conclusions. For example, the U.S. Environmental Protection Agency (EPA) cosponsored an expert panel on neurodevelopmental end points, which concluded that an experimental design used in nine of the studies cited by Dingemans et al. (2011) failed to control for litter effects (Holson et al. 2008).

Although some investigators have set forth the argument that direct dosing of pups precludes the need to control for litter effects, a U.S. EPA cosponsored expert panel (Moser et al. 2005) evaluated this issue and concluded otherwise.

Regardless of whether Dingemans et al. (2011) view the studies by Holson et al. (2008) and Moser et al. (2005) as credible, the authors should have discussed them to some degree. It is understandable that because of space limitations not all studies can be included in a review. However, it was unacceptable to exclude studies that carry the weight of U.S. EPA cosponsored expert panels or other reviews that critically evaluated many of the studies cited by Dingemans et al. (2011) (e.g., Goodman 2009; Hardy et al. 2009; Williams and DeSesso 2010) and came to opposite conclusions.

Although the article by Dingemans et al. (2011) was peer-reviewed, it presents information in a selective, noncritical manner, which is best reserved for public relation pieces communicated in the non–peer-reviewed media.

In the past, M.B. received honoraria totaling $2,000.00 from Albemarle Corporation for his contribution to studies on brominated flame retardants; he received no form of remuneration for this letter. D.S. declares she has no actual or potential competing financial interests.

Marek Banasik
Dominika Suchecka
Institute of Public Health and
Environmental Protection
Warsaw, Poland
E-mail: iphep.banasik@gmail.com

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Neurotoxicity of PBDEs: Dingemans et al. Respond

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Banasik and Suchecka express their discontent with our recent review on the (in-)direct neurotoxic effects of parent and hydroxylated (OH-) polybrominated diphenyl ethers (PBDEs) on the (developing) nervous system (Dingemans et al. 2011). Their main discontent appears to be once more related to the experimental design in a number of cited behavioral studies. However, our aim was to identify and review the mechanisms underlying the observed adverse (behavioral) effects, not to evaluate the experimental design of behavioral studies within a regulatory setting. Nonetheless, approximately 10% of our review was dedicated to describing a number of behavioral studies [12 different studies from seven different research groups, including a 2008 EPA study (Gee and Moser 2008)] that all indicated the occurrence of neurobehavioral effects following developmental exposure to PBDEs. We used this information to create a starting point for the main part of our review of direct and indirect cellular and molecular mechanisms. For readability and space limitations, we were not able to include all studies, concerns, or critiques that have ever been raised. The absence of a citation to a particular study does not mean that we regard it as less credible.

The view that (developmental) exposure to PBDEs induces adverse neurotoxic effects is widely supported by numerous *in vivo*, *ex vivo*, and *in vitro* studies reporting both structural and functional effects (Dingemans et al. 2011). For some time, a lively discussion has been taking place within the scientific community on the experimental design for behavioral developmental neurotoxicity studies for regulatory purposes, in particular considering the statistical unit (Alcock et al. 2011). In short, there is disagreement about whether direct dosing of pups precludes the need to control for litter effects (e.g., Eriksson 2008; Hardy and Stedeford 2008). However, we did not address this topic in our paper because we consider the potential occurrence of a litter effect to be irrelevant for the reviewed cellular and molecular *in vitro* studies, which all indicate that exposure to PBDEs induces neurotoxic effects.

Critical remarks can be found throughout our review (Dingemans et al. 2011), but they are related to cellular and molecular findings, data gaps, or aspects that warrant further investigation. Our main conclusions are related to the specific (developmental) neurotoxic hazard of OH-PBDEs compared with that of their parent congeners via direct neurotoxicity and thyroid disruption. We also pointed out the need to further investigate the impact of active metabolites, concentrations of PBDEs and metabolites in the (developing) brain, and the potentially increased neurotoxic hazard following exposure to mixtures of different environmental contaminants.

Nonetheless, Banasik and Suchecka raise an important issue: the existence of differences in experimental designs for *in vivo* investigation of (developmental) neurotoxicity. Differences exist in the selection of investigated end points and also in methodologies for the investigation of a specific end point, as reviewed for effects on motor activity by brominated flame retardants (Williams and DeSesso 2010). These differences in experi-